ANALYSIS OF NEURODEVELOPMENTAL DISORDER IN SPECIAL CHILD FROM EEG USING MACHINE LEARNING

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ABSTRACT

Angelman syndrome (AS) is a neurodevelopmental disease characterised by learning disability, speech and motor impairments, epilepsy, erratic sleeping habits, and a constitution that overlaps with the syndrome .Electroencephalogram patterns and high-amplitude intermittent delta waves are popular in people with AS. We are looking for a way to quantitatively investigate electroencephalogram architecture in AS. Hybridization classifier was implemented, as well as the suggestion of patients' care as a gym and stress relief level. Children with AS (ages 4–11) and age-matched neurotypical controls had their wake and sleep EEGs analysed retrospectively. To assess long-range and short-range purposeful properties, we measure coherence over many frequencies during wake and sleep.We use both automated and manual methods to quantify sleep spindles victimisation. We incorporate a real-time health monitoring system in this paper.

Keywords: Angelman syndrome, uniparentaldisomy, EEG.

I. INRODUCTION

Developmental delays, a lack of motor deficiency of voice, epilepsy, and an unusual personality phenotype of a positive disposition are all signs of Angelman syndrome (Dan 2008). It's caused by a lack of ube3a gene expression, which can contribute to chromosome 15q11–q13-related disorders. Angelman syndrome is caused by chromosome 15q11-q13 disorders, which are identical to chromosome 15q11-q13 disorders, or Prader-Willi syndrome (a clinically distinct condition, hypotonia, learning difficulties, obesity and hypogonadism) if they relate to chromosome paternal origin, illustrating the phenomenon of genomic imprinting.About 70% of patients, Angelman syndrome is due to a de novo 15q11-q13 micro deletion on the maternally inherited chromosome, which can be detected by fluorescence in situ hybridization (fish). Around 2-3 percent of patients inherit both copies of chromosome 15 from their father but not from their mother, a condition known as paternal uniparental disomy. As a consequence, the mother's usable copy of UBE3A is not inherited. These patients have less extreme phenotypes than deletion patients, such as a greater head circumference, less severe epilepsy, and more words, yet their vocabulary is highly restricted.

Another 3-5 percent of patients do not have a standard maternal DNA methylation history. Patients with uniparental disomy are phenotypically indistinguishable. In 5-10% of patients with a high rate of private de novo mutations, there is a mutation in the maternal UBE3A gene. Finally, up to 10% of typical cases have no cytogenetic or molecular abnormalities [1].Compared to the surprisingly high-risk development of patients with Angelman syndrome, epilepsy can be analyzed for many conditions. Early development of intractable epilepsy is a frequent symptom in children with atypical abs and myoclonic seizures that have a predisposition to seizure-free epileptic status. This may be attributed to a proclivity for hypersynchronous neural activity, which could be related to irregular GABA-mediated transmission due to a loss of ube3a expression or other causes. There has been an increasing awareness of the risk of convulsive disorders in adult patients in recent years [2]. Sleep is essential for the brain's regeneration and cognitive function repair. Sleep is essential for the brain's regeneration and cognitive function repair. Reading, memory consolidation, declarative learning, motor skills, and plasticity have all been shown to support general cognitive ability through sleep spindles [3]. In children and adolescents

with NDD, sleep disorders that are clinically relevant are typical. In children with neurocognitive disabilities, the role of spindle forming on sleep disorders may be exacerbated. As a result, the probability of abnormal sleep spindle forming, as well as sensitivity to it, will be increased by NDD properties [4]. The aim of this review is to explore the relationship between fusiform sleep characteristics and cognitive functions in children with NDD by analyzing and integrating available data on these characteristics. Not only does a clearer understanding of the connection between sleep and NDD provide insight into the pathophysiology and likely management of such conditions, but it also increases our understanding of the link between sleep spindles and child cognitive processes. The properties of the sleep spindle are most certainly a predictor of brain development and function, as well as a glimpse into the underlying mechanisms that keep cognitive behavior going.

SLEEP SPINDLES IN DEVELOPING CHILDREN

The brain's sleep spindles are an oscillatory electrical potential that lasts from one to several seconds and has a frequency of 11-16 Hz (usually 12-14 Hz in healthy adults) [5]. The spindle is classified as a sine wave in electroencephalography of the scalp in hieroglyphic morphology [6] crescendo-de-crescendo (EEG). The number of waveforms per second density, the number of spindle bursts/min NREM sleep, and the last spindle of it are all features of sleep spindles, as is the magnitude of the peak-to-peak variation in the size of the spindle to reflect the voltage of the frequency.

The spindles in newborns sleep for a few seconds are in the low or high alpha or beta frequency range which can be the lack of synchrony in the front middle, which is possible if not coordinated, is possibly due to a lack of brain myelination. The 2-year-old is considered abnormal if the spindle is still asynchronous [7]. Sleep spindles are able to divide into two types based on their frequency and expression in the field, even if they are articulated diffusively in the head in older children and adults and as far as possible the central region and bilateral synchronicity and symmetry modes. Slow spindles (9 to 13Hz) dominate the frontal regions, while fast spindles (>13-16Hz) dominate the central which parietal head regions, and usually precede slow spindles of hundreds of milliseconds. Slow spindles wax and shrink naturally, while fast spindles often shrink. Around the age of two, this difference occurs. Two sleep spindle occupants are thought to emerge, each with a number of generators inside the thalamus and some cortical intervention.

CHILDREN SLEEP SPINDLE WITH NEURODEVELOPMENTAL DISORDER

Regarding the prevalence of sleep disorders in NDD children and the large variety of cognitive deficiencies in this population, research on the role and function of sleep spindles is limited. Some existing studies in children with intellectual disability, ASD, reading disability, and attention deficit hyperactivity disorder (ADHD) have been conducted, but the body of knowledge is mostly out of date, based on non-standard methods, and mostly on the basis of descriptive and correlated research interspersed, typified by disorders or (in some studies), and intellectual performance.

SLEEP SPINDLES IN CHILDREN WITH INTELLECTUAL DISABILITY

Intellectual deficiency is characterized by an IQ of 75 or less, substantial deficiencies in adaptive behavior, and the onset of impairment before the age of 18. The word "mental retardation" was previously used to describe this disease, but it is no longer used. Genetic conditions, disturbances of brain circulation during pregnancy, complications during childbirth, medical problems affecting brain development, and exposure to environmental contaminants are all common causes of mental retardation [10].

Children with NDD have more severe and frequent sleep issues than children with normal development. Insomnia in typically developing infants is often dependent on actions, while insomnia in children

with Down's syndrome is frequently multifactorial, psychological, and medical, and insomnia in NDD is often permanent, lasting until adolescence or adulthood. Wiggs and Storz discovered that these children's sleep issues lasted an average of 7.13 years (SD4.04 years) [14], and Quine discovered that sleep problems lasted longer in children with developmental disabilities [13].

Children with developmental disabilities have alarmingly high rates of sleep disorders: 86 percent in children under the age of 6, 81 percent between the ages of 6 and 11 years [13], and 77 percent between the ages of 12 and 16 years [15]. More than 50 percent of children with NDD under the age of 16 have difficulty waking up and calming down. Quine [13] found that half of children with NDD have sedentary issues which are more than two-thirds of those with night wakefulness have sedentary problems. We were still having issues three years later. ASD is also widespread in children with sleep problems: according to a new survey, 40-85 percent of children with ASD have sleep problems. There are generally long-term sleep delays, including reported sleep disorders, anxiety and frequent sleep after waking at night, and reduced total sleep periods. With ASD aged 2-5 of fifty-3%of controls [19] of 32% compared with at least one sleep problem. ASD children often sleep more than 1 hour to take, a lot of people for 2 to 3 hours an last night Wake you. Sleep problems with ASD tend to persist well past the middle stages of puberty. In the most common sleep problems under the age of 11-13, children with ASD, delayed sleep onset, frequent nighttime wakefulness and reduced sleep time. In the case of ADHD, 70 percent of children with the condition have moderate to extreme sleep issues. The rate is determined by ADHD subtypes, which are most commonly derived from sub sleepiness, which can become more common in blunt subtypes. Furthermore, both medical coma and drug use raise the risk of ADHD sleep disorders. Far more than bedtime resistance, trouble developing, sleep at night, difficulty waking up in the morning, sleep breathing issues, and healthy controls, children and/or parents may not have the sleep problems inherent in ADHD, although the most widely mentioned problem is harder to sleep.

INTELLECTUAL DISABILITY CHILDREN WITH SLEEP SPINDLES AND INTELLECTUAL PERFORMANCE

In this study of shibakaki et al. Functional levels were classified according to the developmental index (DQs) of the participants. The Tsumori questionnaire and the insane questionnaire for infants and young children were carried out, and they were divided into severe, moderate, and slight intellectually handicapped. They discovered that children with intellectual disabilities, including those with intellectual disabilities associated with a variety of disorders (e.g., congenital brain dysplasia, hydrocephalus, Rubinstein-Taybi syndrome, Down syndrome, and chromosomal abnormalities), often had no sleep spindles, and that these children had no sleep spindles with the shorter or no sleep spindles compared to children without intellectual disabilities.

II. SLITERATURE REVIEW

S. E. Goldman, T. J. Bichell [38],Sleep anxiety is more with 20-80 percent of children with Angelman syndrome experiencing a decreased sleep requirement and/or irregular sleep wake periods, this is a typical occurrence. It's unclear what effect these sleep habits have on parental sleep and stress. They discovered that parents with Angelman syndrome had dramatically higher sleep habits and parental stress levels, as measured by structured questionnaires, wrist actigraphy, and polysomnography.ASD is a neurodevelopmental disorder characterized by executive control, vocabulary, emotion, and social function deficits. Pathophysiology in cerebral structural tissues is linked to ASD. We investigated the topographic differences in brain function using absolute, relative, total power, and inferred coherence within and between hemispheres in two children

diagnosed with autistic disorder compared to 20 controls, age and EEG matched to gender, recorded at rest with ocular closure eye, and investigated the quantitative brain wave findings of two children diagnosed with autistic disorder compared to 20 controls, age and EEG matched to gender, recorded at rest with ocular closure eye. The literature survey shown that the Angelman syndrome (AS) was the neurodevelopmental disorder which made the overlap of the phenotype to be the features of intellectual disorder, speech and movement disorder, epilepsy, abnormal sleep, autism.

III. OBJECTIVES OF THE STUDY

The main objectives of proposed system are as follows:

1. Angelman syndrome, UBE3A, collection, sharing as well as exchange of physiological helps to create real time health monitoring system.

2. EEG signals were used to detect the wake and sleep spindles and to predict special children based on associated symptoms.

3. EEG Threshold values are being used to measure Coherence, Spindles, Frequency, and Euclidian distance in order to predict Angelman syndrome in each child or group of children for further comparison.

IV. RESEARCH APPROACH

QUANTITATIVE APPROACH:

- Student's t tests are used to determine overall coherence as a feature of genotype.
- To understand main frequencies ranges (delta, theta, alpha, beta, and gamma) contribute to coherence.
- We compared spectral power, spindle frequency, and spindle length between groups using Student's t tests.
- A two-tailed Fisher's exact test are used to compare trust in manual spindle detection.

QUALITATIVE APPROACH:

- Coherence is being calculated between of 19 EEG electrodes and categorized into short-range and long-range electrode pair's of145 combinations for individuals.
- Coherence is the key to understand the comparisons between children with AS and neurotypical (NT) further it will be measured coherence within frequency bands of interest.
- The purpose of the EEG coherence is to examine separately at the time of wakefulness (NT: 54; AS: 26) and NREM sleep (NT: 54; AS: 13).

V. RESULTS AND DISCUSSION

The primary goal of this research is to develop a real-time health monitoring system for Angelman syndrome patients that allows for the collection, sharing, and exchange of physiological data. The framework is developed on the Windows platform with Ubuntu. PyCharm is a development environment.

- **DATASET USED**

  The system uses ClinicalTrials.gov identifier: NCT00296764 as a dataset.

- **PROPOSED SYSTEM ARCHITECTURE**

  The figure 3.1 indicates the proposed system architecture which is being analyzed retrospective clinical EEGs from children with a genetically confirmed diagnosis of AS and age matched neurotypical controls.

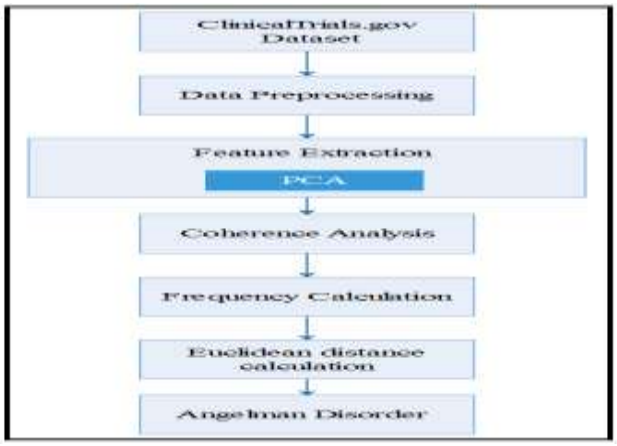

Fig. 1 System Architecture

- **DATASET:** ClinicalTrials.gov identifier: NCT00296764.
- **DATA PREPROCESSING:** AS EEGs is pre-processed in tandem using identical techniques to reduce the intrinsic effect of matching recordings from two locations. The pre-processing process included re-referencing signals to connected ears, filtering, sleep/wake labelling, and artefact elimination.
- **FEATURE EXTRACTION:** Electroencephalography (EEG) has long been used to diagnose a variety of heart conditions. The EEG is a practical representation of the position and degree of electrical commotion generated by depolarization and repolarization of the atria and ventricles-QRS-T waves make up one cardiac cycle in an EEG signal. The EEG feature extraction method extracts basic features (amplitudes and intervals) that can then be used in automated analysis. For feature extraction, the framework employs the principal component analysis (PCA) algorithm.
- **COHERENCE ANALYSIS:** Due to sleep or wake coding and artefact elimination, pre-processed EEG signals is non-continuous and non-uniform. As a result, we measured coherence for each segment of pre-processed data separately, then summed coherence across segments when weighting segment volume. For coherence tests, we only used continuous data that was longer than 10s.
- **FREQUENCY CALCULATION:** As tested during wakefulness and sleep, children with AS had increased long-term EEG coherence over a wide range of frequencies. While sleeping, children with AS had higher long-range EEG coherence, especially in the gamma band. In AS children's EEGs, sleep spindles were less frequent, and these spindles were shorter in duration than in neurotypical children's EEGs.
- **MEASURE OF EUCLIDEAN DISTANCE**: The electrode pairs can be divided into short-range and long-range pairs based on Euclidean distance. Because of the possible confound of volume conduction, electrodes directly adjacent to each other were excluded from the study.
- **ANGELMAN DISORDER:** Detection of Angelman Disorder.

IV. DISCUSSION

The aim of this paper is to examine and integrate (as far as possible) the association between cognitive performance and available evidence of sleep spindle characteristics in children with NDD. We should be aware of the crucial methodology weaknesses of current work before attempting to integrate the results through various obstacles. Many of the replicated studies had limited sample sizes and even overestimated the magnitude of the effect. This is made more complicated if the researchers published data from the same

participants several times. The sample's heterogeneity is another drawback. First, each of the NDDs discussed has its own clinical heterogeneity. Heterogeneity can also be raised from other information levels and/or the inclusion of comorbid status participants. This kind of causes the results to other settings and circumstances that can be generalized about whether question. Other problems are difficult to directly compare the results can be a research of methodological differences.

For e.g., some studies reported brainwave patterns during the night, while others were induced by chloral hydrate during the day, with normal clinical electroencephalography or, in the latter case, where the drug effect could not be eliminated, the duration of time spent in stage 2NREM sleep was reduced, and less sleep spindle was detected. The study of sleep spindles and comprehension in children with NDD may be hampered by these limitations. It may be difficult to achieve a bigger, more homogeneous population due to the prevalence of disorders and their clinical existence. Cognitive research, as a realistic problem, is a major obstacle to the technical and financial viability of large-scale, homogeneous trials that use quantitative tests of sleep and cognition. The electrodes can be painful, and being in a new environment can be terrifying, and cognitive research, as a realistic concern, is a real obstacle to large-scale, homogeneous trials that use quantitative measures of sleep and cognition. Our study collects and analyses information on sleep spindle characteristics in children with NDD in order to link these differences to cognitive processes.

VI. CONCLUSION

In children with AS, the analysis provided the largest objective data collection of sleep quality parameters. The standard of sleep among children as a sleep condition identified by parents and at night for individual children was distinguished by broad variation. The questionnaire report of parents over the seven-night cycle emphasises this inconsistency, because unlike those found in children with a normally emerging on a weeknight, the change in overall sleep time on children with AS is attributed to a lack of environmental and social constraints.

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